1,2-DIBROMO-3-CHLOROPROPANE

1,2-Dibromo-3-chloropropane is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 96-12-8 $BrCH_2BrCHCH_2Cl$

Molecular Formula: $C_3H_5Br_2Cl$

1,2-Dibromo-3-chloropropane (DBCP) is a dark amber-to-dark brown liquid that has a pungent odor. It is slightly soluble in water, and miscible in aliphatic and aromatic hydrocarbon solvents (NTP, 1991).

Physical Properties of 1,2-Dibromo-3-chloropropane

Synonyms: DBCP; dibromochloropropane; 3-chloro-1,2-dibromopropane; OS1897; Fumazone; Nemafume; Nemagon

Molecular Weight:	236.36
Boiling Point:	196 ^oC
Melting Point:	6 ^oC
Flash Point:	76.6 ^oC (170 ^oF) open cup
Vapor Density:	2.09 at 14 ^oC (air = 1)
Density/Specific Gravity:	2.08 at 20/20 ^oC (water = 1)
Vapor Pressure:	0.8 mm Hg at 21 ^oC
Log Octanol/Water Partition Coefficient:	2.43
Water Solubility:	1,230 mg/L at 20 ^oC
Henry's Law Constant:	1.47 x 10^{-4} atm-m^3/mole
Conversion Factor:	1 ppm = 9.7 mg/m^3

(Howard 1990; Merck 1983; Sax 1987; U.S. EPA, 1994a)

SOURCES AND EMISSIONS

A. Sources

DBCP is used as a laboratory reactant, intermediate in organic synthesis, and commercial preparation for the flame retardant tris(2,3-dibromopropyl)phosphate (Howard, 1990).

DBCP was registered for use as a pesticide, however prior to January 1, 1980, it was no longer registered for pesticidal use in California (DPR, 1996).

B. Emissions

No emissions of DBCP from stationary sources in California were reported, based on data obtained from the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

DBCP is not known to occur as a natural product (HSDB, 1991).

AMBIENT CONCENTRATIONS

No Air Resources Board data exist for ambient measurements of DBCP. However, the United States Environmental Protection Agency (U.S. EPA) reported a mean concentration of 0.01 micrograms per cubic meter ($\mu g/m^3$) or 0.001 parts per billion at one U.S. urban location in 1976 (U.S. EPA, 1993a).

INDOOR SOURCES AND CONCENTRATIONS

No information about the indoor sources and concentrations of DBCP was found in the readily-available literature.

ATMOSPHERIC PERSISTENCE

The dominant chemical loss process for DBCP is by reaction with the hydroxyl (OH) radical. The calculated half-life and lifetime of DBCP due to reaction with the OH radical are 23 days and 33 days, respectively (Atkinson, 1995). The formation of 1,2-dibromopropanol and chlorobromopropanol may be expected from this reaction (Kao, 1994).

AB2588 RISK ASSESSMENT INFORMATION

Since no emissions of DBCP from stationary sources in California have been reported under the AB 2588 program, it was not listed in any of the risk assessments reviewed by the Office of Environmental Health Hazard Assessment.

HEALTH EFFECTS

Probable routes of human exposure to DBCP are inhalation, ingestion, and dermal contact.

Non-Cancer: DBCP is a moderate central nervous system depressant and an eye, skin, and respiratory tract irritant. Acute overexposure to DBCP in humans by inhalation may cause central nervous system effects, with symptoms including drowsiness, narcosis, and pulmonary congestion. Chronic inhalation exposure has been reported to cause kidney and liver effects in rats and mice (U.S. EPA, 1994a).

A chronic non-cancer Reference Exposure Level (REL) of 0.2 $\mu g/m^3$ is listed for DBCP in the California Air Pollution Control Officers Association Air Toxics "Hot Spots" Program, Revised

1992 Risk Assessment Guidelines. The toxicological endpoints for chronic toxicity are the respiratory, reproductive and gastrointestinal systems, and liver (CAPCOA, 1993). The U.S. EPA has established a Reference Concentration (RfC) for DBCP of 0.2 $\mu g/m^3$ based on testicular effects in rabbits. The U.S. EPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic non-cancer effects. The U.S. EPA has not established an oral Reference Dose (RfD) (U.S. EPA, 1994a).

Chronic occupational exposure to DBCP caused decreased sperm counts in men; however, no association between paternal exposure and birth defects, prematurity, mortality, or spontaneous abortions was noted. Testicular effects and decreased sperm count were observed in rabbits chronically exposed to DBCP by inhalation (U.S. EPA, 1994a). Also, the State of California has determined under Proposition 65 that DBCP causes male reproductive toxicity (CCR, 1996).

Cancer: Human data on the cancer potential of DBCP are inadequate according to the U.S. EPA. In animal studies, rats and mice exposed by inhalation developed nasal tumors. The U.S. EPA has classified DBCP in Group B2: Probable human carcinogen (U.S. EPA, 1994a). The International Agency for Research on Cancer (IARC) has classified DBCP in Group 2B: Possible human carcinogen, based on sufficient evidence in animals and inadequate data in humans (IARC, 1987a).

The State of California has determined under Proposition 65 that DBCP is a carcinogen (CCR, 1996). The inhalation potency factor that has been used as a basis for regulatory action in California is 2×10^{-3} (microgram per cubic meter)$^{-1}$ (OEHHA, 1994). In other words, the potential excess cancer risk for a person exposed over a lifetime to 1 $\mu g/m^3$ of DBCP is estimated to be no greater than 2,000 in 1 million. The oral potency factor that has been used as a basis for regulatory action in California is 7.0 (milligram per kilogram per day)$^{-1}$ (OEHHA, 1994).